member in humans of a family of proteins called cathelicidins. LL–37/hCAP18 is produced by neutrophils and various epithelial cells. LL–37 is well known as an antimicrobial peptide. However, although antimicrobial peptides have generally been considered to contribute to host innate antimicrobial defense, some of them may also contribute to adaptive immunity against microbial infection. The inventors have shown that LL–37 utilizes formyl peptide receptor-like 1 (FPLR1) as a receptor to activate human neutrophils, monocytes, and T cells. Since leukocytes participate in both innate and adaptive immunity, the fact that LL–37 can chemoattract human leukocytes may provide one additional mechanism by which LL–37 can contribute to host defense against microbial invasion, by participating in the recruitment of leukocytes to sites of infection. The invention claims methods of enhancing immune responses through the administration of LL–37 alone, in conjunction with a vaccine, and methods of treating autoimmune diseases. The invention is further described in Chertov *et al.,* ''LL–37, the neutrophil granule-and epithelial cell-derived cathelicidin, utilizes formyl peptide receptor-like 1 (FPRL1) as a receptor to chemoattract human peripheral blood neutrophils, monocytes, and T cells,'' J Exp. Med. 2000 Oct 2;192(7):1069–74.

A Method for Bioconjugation Using Diels-Alder Cycloaddition

Vince Pozsgay (NICHD)

DHHS Reference No. E–126–00/0 filed 09 Aug 2000

This invention relates to a new method for the synthesis of conjugate vaccines using the Diels-Alder cycloaddition reaction to covalently attach a carbohydrate antigen from a pathogen to a protein carrier. The Diels-Alder reaction has not been extended to conjugation involving biopolymers or other types of polymeric materials. Advantages of this method are that cross-linking during conjugation is entirely avoided in addition to the mild chemical conditions under which this synthesis method proceeds. Diels-Alder reactions commonly take place in high-temperature environments; the method contemplated by this invention takes place at much lower temperatures. In addition to claiming methods of synthesis for conjugate vaccines using the Diels-Alder cycloaddition, the patent application claims vaccines produced utilizing the method, and methods of inducing antibodies which react with the polysaccharides contemplated by the invention.

5-Substituted Derivatives of Conformationally Locked Nucleoside Analogues

Victor Marquez, Pamela Russ (NCI)

DHHS Reference No. E–249–00/0 filed 26 Jul 2000

This invention relates to 5-substituted derivatives of conformationally locked nucleoside analogues and methods of using these derivatives as antiviral and anticancer agents. The compounds contemplated by the invention are nucleoside analogues where the 5-substituent is a halogen, alkyl, alkene, halovinyl or alkyne group, and the nucleotide base is cytosine or uracil. The analogues are particularly effective in treating viral infections, specifically infections of DNA viruses such as Herpes simplex virus (HSV), Varicella zoster virus (VSV), Epstein Barr virus (EBV), and Cytomegalovirus (CMV) as well as members of the Poxviridae family. The inventors have demonstrated in plaque reduction assays that 5-substituted uracils (bromo, iodo, and bromovinyl) attached to a bicyclo[3.1.0]hexane template are thirty times more potent than acyclovir against HSV–1 and HSV–2.

Bacteriophage Having Multiple Host Range

Carl Merril (NIMH), Sankar Adhya (NCI), Dean Scholl (NIMH)

DHHS Reference No. E–257–00/0 filed 25 Jul 2000

Recently, there has been a renewed interest in the use of phages to treat bacterial infections. The inventors have discovered FK1–5, a highly lytic, non-lysogenic, stable bacteriophage with the ability to kill bacteria rapidly, making it a good candidate for phage therapy. The designation FK1–5 denotes the phage's ability to infect E. coli strains that contain the K1 polysaccharide in their outer capsule as well as E. coli strains that contain the K5 polysaccharide in their outer capsule. Sequence analysis of the tail proteins of phage FK1–5 by the inventors has shown that they are arranged in a cassette structure, suggesting that the host range of phages can be broadened to other K antigens, and even possibly other species of bacteria by recombinant techniques. FK1–5 has a particular advantage because it recognizes and attaches to the structures that confer virulence to bacteria. The inventors' demonstration that a phage can contain multiple tail proteins that expand its host range is useful for generating phage with broad-spectrum antibacterial properties for the treatment of infectious diseases. The inventors have completed in vitro studies on this phage. Furthermore, because of the possibility of engineering the expression of recombinant tail proteins, gene transfer to organisms that are not normally infected by phages is also contemplated by the invention.

Dated: December 4, 2000.

Jack Spiegel,

Director, Division of Technology, Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. 00–31525 Filed 12–11–00; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, Public Health Service, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by writing to the indicated licensing contact at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7057; fax: 301/402–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

A Mouse Model of UV-Inducible Cutaneous Malignant Melanoma

Glenn Merlino *et al.* (NCI)

DHHS Reference No. E–281–00/0

Licensing Contact: Elaine White; 301/496–7056 ext. 282; e-mail: gesee@od.nih.gov

The current invention embodies a genetically engineered mouse harboring a hepatocyte growth factor/scatter factor transgene (''HGF/SF''). The Met signaling pathway, which has been implicated in the development of human melanoma, is chronically

activated in the HGF/SF mice. Upon exposure to a single neonatal dose of erythrogenic UV radiation, the mice develop cutaneous malignant melanoma which is consistent with the epidemiology and pathogenesis of melanomas observed in humans. The mice, therefore, represent a valuable model for studying the development of malignant melanoma in humans, for determining the consequences of ultraviolet radiation, and for assessing the efficacy of therapeutic agents and vaccines against melanoma. While no patent rights are available for this invention, breeding pairs of mice are available for licensing via Biological Materials License Agreements.

Gamma-Glutamyl Transpeptidase Inhibitors: Novel Chemotherapeutic Agents

Robert E. London, Scott A. Gabel (NIEHS)
DHHS Reference No. E–243–00/0 filed 05 Oct 2000
Licensing Contact: Richard Rodriguez; 301/496–7056 ext. 287; e-mail: rodrigur@od.nih.gov

Gamma-glutamyl transpeptidase (GGTP) plays a central role in the metabolism of glutathione. It has been shown to be a marker for neoplasia and cell transformation, and it is induced by the presence of many anti-cancer drugs. Common human epithelial tumors, including, but not necessarily limited to, breast, ovarian and prostate tumors are GGTP-positive. The invention relates to novel inhibitors of GGTP, and their use to treat cancer. In particular, the technology could be used to (1) interfere with glutathione metabolism in GGTP-positive cancers by perhaps altering the cellular orientation of GGTP; (2) potentiate the effects of radiation and chemotherapeutic drugs, in particular, cisplatin, on cancer cells by interfering with cysteine recycling and glutathione regeneration; and (3) reduce renal toxicity for some chemotherapeutic drugs by blocking the metabolism of glutathione-conjugates into toxic agents, *e.g.,* mercapturic acids. The patent application contains composition of matter claims as well as method claims.

Protein Kinase A and the Carney Complex

Constantine A. Stratakis, Lawrence S. Kirschner (NICHD)
DHHS Reference No. 259–00/0 filed 25 Aug 2000
Licensing Contact: Richard Rodriguez; 301/496–7056 ext. 287; e-mail: rodrigur@od.nih.gov

The present invention provides compositions and methods useful in the diagnosis and prognosis of Carney complex (CNC), as well as methods and compositions for the identification of compounds useful in the treatment and/or prevention of CNC. CNC is a multiple endocrine neoplasia syndrome that affects the adrenal cortex, pituitary gland, thyroid gland and gonads. Additionally, compositions and methods are provided for the diagnosis and treatment of conditions associated with skin pigmentation defects, including but not limited to, freckling, as well as endocrine tumors including, but not limited to, adrenal and pituitary tumors. Finally, compositions and methods are provided for the diagnosis and treatment of various types of cancers associated with abnormal protein kinase A activity, and cancers and tumors in which protein kinase A regulatory subunit 1A acts as a tumor-suppressor gene. These actions are possible due to the identification of specific genetic sequences, and the use of this information in assay systems to detect, diagnose and treat the aforementioned conditions.

SH2 Domain Binding Inhibitors

Terrence R. Burke, Yang Gao, Johannes Voight (NCI)
DHHS Reference No. E–262–00/0 filed 22 Aug 2000
Licensing Contact: Richard Rodriguez; 301/496–7056 ext. 287; e-mail: rodrigur@od.nih.gov

Signal transduction, the process of relaying extracellular messages to the intracellular cytoplasm and the nucleus, is critical to normal cellular homeostasis, and protein-tyrosine kinases play a central role in this biological function. Examples of this latter class of enzymes include the PDGF receptor, the FGF receptor, the HGF receptor, members of the EGF receptor family, including the EGF receptor itself and erb–B2, erb–B3 and erb–B4 kinases; the src kinase family, Fak kinase and the Jak kinase family. Protein-tyrosine phosphorylation is known to be involved in modulating the activity of a variety of target enzymes and in the formation of specific complex networks involved in signal transduction via proteins containing specific amino acid sequences, called the Src homology 2, or SH2 domain. A malfunction in this protein-tyrosine phosphorylation through tyrosine kinase overexpression and/or deregulation, can be manifested by various oncogenic and hyperproliferative disorders, such as cancer, inflammation, autoimmune disease, hyperproliferative skin disorders, *e.g.,* psoriasis and allergy/asthma. The disclosed compounds, *e.g.* peptides, preferably, macrocyclic peptides, are SH2 domain inhibitors with enhanced binding affinity. The claims of the current application are directed to compositions of matter and methods of use which provide for the diagnosis, testing and treatment of the aforementioned disease states.

Use and Targeting of CD98 Light-Chain Proteins in Therapies for Thyroid Hormone Disorders

Yun-Bo Shi (NICHD)
DHHS Reference No. E–054–00/0 filed 30 Jun 2000
Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: shinnm@od.nih.gov

Thyroid hormone disorders are among the most common problems in the Western world. These include hypo-and hyper-thyroidism (including goiter), as well as obesity and developmental abnormalities caused by excess or deficient levels of thyroid hormones during pregnancy.

The NIH announces the discovery of a protein, which is a member of the CD98 light-chain permease family, which acts as a thyroid hormone transporter across vertebrate cell membranes. This protein provides a missing link in the chain by which thyroid hormones in the blood reach the cell nucleus. By utilizing the CDNA of this protein, genomic libraries can be screened for sequences capable of being used as primers for use in diagnostics. Also, by targeting this protein through drug discovery, new treatments for thyroid disorders may be found and developed.

Method of Regulating Interleukin-12 (IL–12) Production by Administering CCR5 Agonists and Antagonists

Sher *et al.* (NIAID)
PCT/US00/01019 filed 14 Jan 2000
Licensing Contact: J.P. Kim; 301/496–7056 ext. 264; e-mail: kimj@od.nih.gov

Interleukin-12 (IL–12) is a cytokine produced by the body which is necessary for the development of effective cellular immunity against many microbial agents. Increasing IL–12 production has been shown to both enhance the immune clearance of microbial agents as well as augment the protection induced by vaccines. At the same time a number of inflammatory diseases are associated with the excess production of this cytokine. Therefore, methods are needed to both boost IL–12 production for the induction of host resistance as well as suppress it to treat these immunopathologic disorders.

The present invention relates to methods for increasing IL–12

production in a cell by administering CCR5 agonists and methods for decreasing IL–12 production in a cell administering CCR5 antagonists. The invention also relates to methods for increasing IL–12 production by administering CCR5 agonists and to methods for decreasing IL–12 production in a subject by administering CCR5 antagonists.

Dated: November 11, 2000.

Jack Spiegel,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. 00–31526 Filed 12–11–00; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Mental Health; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the following meetings.

The meetings will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Institute of Mental Health Special Emphasis Panel.

Date: December 7, 2000.

Time: 9:00 a.m. to 10:30 a.m.

Agenda: To review and evaluate grant applications.

Place: Neuroscience Center, National Institutes of Health, 6001 Executive Blvd., Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: Henry J. Haigler, PhD, Scientific Review Administrator, Division of Extramural Activities, National Institute of Mental Health, NIH, Neuroscience Center, 6001 Executive Blvd., Rm. 6150, MSC 9608, Bethesda, MD 20892–9608, 301/443–7216.

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

Name of Committee: National Institute of Mental Health Special Emphasis Panel.

Date: December 7, 2000.

Time: 10:30 a.m. to 11:00 a.m.

Agenda: To review and evaluate grant applications.

Place: Neuroscience Center, National Institutes of Health, 6001 Executive Blvd., Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: Henry J. Haigler, PhD, Scientific Review Administrator, Division of Extramural Activities, National Institute of Mental Health, NIH, Neuroscience Center, 6001 Executive Blvd., Rm. 6150, MSC 9608, Bethesda, MD 20892–9608, 301/443–7216.

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

(Catalogue of Federal Domestic Assistance Program Nos. 93.242, Mental Health Research Grants; 93.281, Scientist Development Award, Scientist Development Award for Clinicians, and Research Scientist Award; 93.282, Mental Health National Research Service Awards for Research Training, National Institutes of Health, HHS)

Dated: December 4, 2000.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 00–31518 Filed 12–11–00; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Nursing Research; Notice of Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the National Advisory Council for Nursing Research.

The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and/or contract proposals and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications and/or contract proposals, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Advisory Council for Nursing Research.

Date: January 23–24, 2001.

Open: January 23, 2001, 1:00 p.m. to 5:00 p.m.

Agenda: For discussion of program policies and issues.

Place: 9000 Rockville Pike, Building 31, Conference Room 10, Bethesda, MD 20892.

Closed: January 24, 2001, 9:30 a.m. to 1:00 p.m.

Agenda: To review and evaluate grant applications and/or proposals.

Place: 9000 Rockville Pike, Building 31, Conference Room 10, Bethesda, MD 20892.

Contact Person: Mary Leveck, PhD, Associate Director for Scientific Programs, NINR, NIH, Building 31, Room 5B05, Bethesda, MD 20892, (301) 594–5963.

(Catalogue of Federal Domestic Assistance Program Nos. 93.361, Nursing Research, National Institutes of Health, HHS)

Dated: December 4, 2000.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 00–31519 Filed 12–11–00; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Mental Health; Notice of Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the National Advisory Mental Health Council.

The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Advisory Council.

Date: January 18–19, 2001.

Closed: January 18, 2001, 10:30 a.m. to recess.

Agenda: To review and evaluate grant applications.